züge der physiologischen Psychologie. Translation, to a man capable of good original work, is a heavy and sometimes unjustifiable sacrifice, but Wundt easily accessible to any one who can read English is something worth considerable sacrifice and something for which the translator's colleagues will not fail to be grateful.

That we have not had a translation long ago is not Prof. Titchener's fault. In 1890-91 he had already completed one of the third edition, but not before a fourth was in prospect. In 1899 again a complete translation of the fourth edition was given over in view of the present fifth edition. The translation now published is a fresh one of the entire work, the others having been wholly discarded. Such an experience is hard for the translator, but gets its reward in the quality of the final product.

The original fifth edition of the Grundzüge consists of three volumes (pp. 553, 686, 796) and an index (pp. 134). The first volume of the translation covers 338 pages of Vol. I of the original, embracing the "Introduction" and "Part I: On the Bodily Substrate of the Mental Life," together with a ten-page section on "Pre-psychological Concepts" which is found in the fourth edition but not in the fifth, and a volume index of names and subjects.

As a matter of course the work has been carefully and conscientiously done. In point of training, experience and sympathy Prof. Titchener is uniquely fitted for his task. Translator's footnotes have been added sparingly: in some cases for the special rendition of difficult terms, in others for references to English versions of works cited (*e. g.*, to Jowett's Plato), in still others for bringing the statements of the text into harmony with recent discoveries in embryology and neurology. Perhaps the utter impossibility of turning Wundt's German into intelligent English by a literal translation, perhaps the enforced revisions of the English version, perhaps the translator's veteran skill, perhaps all of them together, have made the style less angular than that of Külpe's manual some years ago, and therefore much pleasanter reading—a point of no small importance in a work of this kind.

It is hardly necessary to add that the psychologist who has not already facile use of a copy of the original should possess himself of the translation, even if he should have to suspend his subscription to the *American Journal* to purchase it! E. C. S.

PSYCHIATRY.

DR. ISADOR H. CORIAT.

RECENT LITERATURE ON THE PATHOLOGY OF DEMENTIA PRÆCOX.

The feeling that there must exist an anatomical basis for the production of various psychoses, will perhaps explain the recent studies devoted to this end in dementia præcox. Kahlbaum has already reported at length the results of seven autopsies in katatonia, in which the microscopic examination was negative, while Alzheimer and Nissl in the same disease found a neuroglia increase, especially in the deeper layers of the cortex. W. R. Dunton (American Journal of Insanity,

Jan., 1903), made an exhaustive examination of the central nervous system in a case of the katatonic form of dementia præcox of four years' duration, with death from pulmonary tuberculosis. The cranial bones were thickened and the dura free. The cortex was firm and of even consistency, but in the right hemisphere the episylvian sulcus was found to be markedly developed, while the paracentral convolutions were noticeably narrow. Sections from the frontal, temporal, paracentral, Broca's, the anterior and posterior central convolutions, cuneus, hypophysis, angular gyrus, caudate and lenticular nuclei, facial area, cerebellum, lumbar and dorsal cord and the spinal ganglia disclosed the following findings. Slight cell changes were found everywhere, being most marked in the first frontal convolution. These changes consisted of central chromatolysis, a slight degree of pale yellow pigmentation, slight cell atrophy, dislocation and swelling of the nucleus and folding of the nuclear membrane. The deeper layers seemed to be mostly affected. Phagocytosis was well marked and there was slight increase of neuroglia nuclei, but no changes in the medullated fibres and no marked vascular changes. Drawing his conclusions from the study of this one case, the writer says, "The positive findings are suggestive and seem to me to indicate the lines upon which further research can be best carried out for future study of this class of cases."

Dunton's second paper (American Journal of Insanity, April, 1904) relates to the case of a female, aet. 47, with a tubercular heredity. There was increasing dementia and rapid and extreme emaciation, the weight in 8 months decreasing from 180 to 90 lbs. A few days before death there were noticed muscular movements, general coarse tremor and at times clonic movements of the extremities. The dura was adherent over the region of the first frontal convolutions; the brain was symmetrical, the convolutions somewhat atrophied and there was engorgement of the vessels. The specimens for histological examination were taken from the same regions as in the first case. The cells in the cortex showed the following changes; increased pale yellow pigment, frequent central chromatolysis, cell often swollen, atrophied, displaced and distorted nuclei. In the cerebellum, there was a decrease of the Purkinje cells, which were distorted and atrophied and had a granular appearance, while the nuclei were distorted. "Ghost cells" were present. There was an increase of the tangential fibres; the neuroglia sections were unsuccessful, but an increase of neuroglia nuclei was observed by other stains. In using the polychrome stain, the white matter on decolorization, retained its color longer than the cortex, and this indicated to the author some unusual chemical change in the brain.

From his study of these two cases, and from the literature reviewed in the papers, the author considers that the pathology of dementia præcox is being established. The nerve cells in the cortex diminish in dementia as in idiocy, with possible changes in their orientation. He inclines to the hazy auto-intoxication hypothesis, as the possible etiological factor concerned in these changes. These chromatolytic cell changes described by Dunton are by no means constant or pathognomonic for dementia præcox. They have been found in a multitude of other conditions; general paralysis, delirium tremens, facial paralysis, Landry's paralysis, alcoholic neuritis, tetanus, typhoid fever, pellagrous insanity, phosphorus poisoning, fish poisoning with grave gastro-intestinal symptoms, carcinoma in the vagus nucleus, in diphtheria, and in various terminal delirious and depressive states. This last, when associated with emaciation, diarrhea, cachexia, motor weakness and twitchings or rigidity, may form the terminal complex of any primary mental disorder. It may occur in dementia præcox, but is found

especially in those psychoses around the climacteric period, and this end symptom-complex is termed central neuritis by Dr. Adolf Meyer. The cell changes in these cases (chromatolysis, "axonal reaction"), are associated with degeneration of their corresponding sets of fibres. In both of Dunton's cases there was extreme emaciation, while in the second, there was noted tremor and clonic movements of the extremities a few days before death. The cell changes in both these cases, therefore, are in harmony with the clinical and anatomical picture of central neuritis; the process is a terminal one merely, neither the result nor the underlying cause of the psychosis itself, but modified by an intercurrent disorder. In one of our own cases of typical central neuritis, a careful examination of the urine, both chemically and physiologically, failed to reveal any toxic products.

Lubouchine (Modifications anatomo-pathologiques de l' ecorce cerebrale dans deux cas de Démence Precoce, Journal de Neuropathologie et de Psychiatrie du nom S. S. Korsakoff, 1902, livre 1-2. P. 61-72) in two cases of dementia præcox finds atrophy, and pigmentary and fatty degeneration of the cells, some disappearance and degeneration of the tangential and subcortical fibres, enlargement of the peri-vascular spaces, proliferation of neuroglia in the molecular layer, in the layer of the small pyramidal cells and around the blood-vessels.

Bridier (Essai sur l'Anatomie Pathologique des Démences. Thése de Lyon, 1902), states that in whatever form of dementia, the lesions in the cortex are practically the same, and involve the nerve cells, the supporting tissue and the vessels. These lesions consist of fatty and pigmentary degeneration, central chromatolysis, division of the cell nucleus, and later atrophy of the cell body. There was also proliferation of the neuroglia in the layer of pyramidal cells, appearance of migratory elements and arterio-sclerosis of the blood vessels.

Klippel and L' Hermitte (Démence Precoce, Anatomie Pathologique et Pathogénie, Revue de Psychiatrie et de Psychologie Experimentale. Fev., 1904. No. 2) examined four cases of dementia præcox for the size of the cell bodies, drawing and measuring a fixed number of pyramidal cells in the motor zones and association centres. They found some anomalies of the fissures, absence of vascular changes and no neuroglia increase, the cells of the frontal cortex and the pyramidal cells were smaller than normal and showed marked chromatolysis and pigmentation with dislocation of the nucleus. In two cases assymetry of the cerebellum was noted. The atrophy of the cells was limited mostly to the association centres, and the various changes were looked upon as the result of long continued but rather weak pathogenic agents, of an unknown nature.

It seems to us that these few cases are far from establishing any definite anatomical basis for dementia præcox, and do not explain the characteristic symptomatology of this disease. Besides, the group is a wide one and there is some uncertainty whether we are dealing with a clinical entity, and further observation may establish independent subdivisions. Until then our efforts are premature, we are not justified in drawing an anatomical conclusion, or endeavoring to establish an organic basis for the disease. The recent isolation of a specific micro-organism from the blood in katatonia, needs only to be mentioned, to perceive it's absurdity. Possibly physical chemistry or the application of the side-chain theory may throw some light on the question.

GANSER'S SYMPTOM.

This is one of the interesting questions of contemporary psychiatry. Ganser first published his observations on random replies in 1897, and since then there has arisen much discussion as to whether these replies are of a hysterical or katatonic nature. Ganser looked upon

it as a hysterical symptom, Raecke thought it to be merely a stigma of hysteria, Nissl as katatonic negativism, while more recently Henneberg (Ueber das Gansersche Symptom.—Allg. Zeit. f. Psychiatrie. Bd. LXI, H. 5, 1904), in a record of thirteen cases showing absurd replies found that the syndrome occurred not only in hysterical stupor and dreamy states, but also in manic conditions, melancholia and dementia præcox. The most striking thing is the absolute absurdity of the replies, even to the most simple questions, the names of familiar objects or in simple calculation. The patient listens attentively, understands the question, tries hard to give a correct answer, but in spite of himself the replies astonish us by their absurdity. A few details of a case from Henneberg's series (Case 13), may perhaps serve to make this more clear. How many legs has a dog? "Five legs." A horse? "Also five." A crow? "I have not seen any." A sparrow? "It has also four feet." 2×2?="5." 2×2?="6." 2×5?="8."

Soukhanoff has given a very excellent résumé of the entire question (Sur la Syndrome de Ganser ou de Symptomo-Complexus des Reponses Absurdes, Rev. Neurol., Ann. XII, No. 17. Sept. 15, 1904). According to him, this syndrome differs from cases of mental confusion, where the replies are absurd, in that they are few in number. He believes it to be part of the category of hysterical disorders, but believes it may also accompany dementia præcox. Cases which show the syndrome do not terminate by recovery, but evolve towards a psychic state of doubtful prognosis, not necessarily deterioration, as no case of hysteria deteriorates. It is an associational disorder, and when the patient starts to reply, ideas of another order suddenly surge up in his consciousness. In hysterical paralysis, one often observes a loss of function of isolated muscles, simulating an organic lesion, and in Ganser's syndrome we have a partial analogue, but it takes place in the association apparatus, it is a partial memory disorder, a disorder of the superior logical mechanism. The question of simulation in medico-legal cases, where random replies are given in an otherwise connected production, is also of special importance.

Description of the Brains and Spinal Cords of Two Brothers. Dead of Hereditary Ataxia. (Cases XVIII and XX of the series in the family described by Dr. Sanger Brown with a Clinical Introduction by Dr. Sanger Brown.) By LEWELLYS F. BARKER. Decennial Publications of the University of Chicago, 1903.

The clinical report of the series was first published in 1892, and two of the histories are here reproduced in detail with the symptoms that have since appeared. In addition, a short résumé of the symptomatology of the disease is given. The anatomical findings, especially the description of the convolutions and sulci of the two brains, are given in great detail, not only on account of the rarity of the condition, but as the cases afforded an excellent opportunity for the topographical comparison of the brains of two brothers. The summary of these two cases shows the brains and cords to have been relatively small, the cerebral cortex was well fissured and showed no deviations from the normal type. In the cord, measurements showed an abnormal ratio between the areas of gray and white matter, as revealed in cross sections. There was a marked degeneration of the gray and white matter of the spinal cord, medulla and cerebellum and involved chiefly the cells and fibres of the centripetal paths. In the cord, there was an increase in glia tissue. It is hoped in a future paper to enter more fully into a discussion of the relation of the lesions to the clinical symptoms. The paper is illustrated with twelve plates, comprising in all forty-six figures of great typographical excellence.